IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant	:	Bertram L. Jacobs et al.			
Serial No.	:	09/837,998	Examiner	:	Guzo, Davis
Filed	:	April 19, 2001	Group Art Unit	:	1636
For	:	VIRAL VECTORS HAVING REDUCED VIRULENCE			

DECLARATION UNDER 37 C.F.R. § 1.132

I hereby certify that this paper is being deposited with the United States Postal Service as first class mail in an envelope addressed to: Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450

October 14, 2003
Date of Deposit

Carmella L. Stephens	41,328
Attorney/Agent Name	PTO Registration No.
Carmella L. Stephens	October 10, 2003
Signature	Date of Signature

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Dear Sir:

I, Bertram Jacobs of 1004 S. Wilson St., Tempe, AZ 85281, hereby declare as follows:

1. I am a joint inventor of the invention described and claimed in the above-captioned patent application ("the present invention"), and hold the position of Professor at the Arizona State University in Tempe, Arizona. A copy of my Curriculum Vitae is attached as Exhibit A.

2. The present invention provides recombinant vaccinia virus having a deletion of the region encoding the C-terminal seven amino acids of the E3L gene product (E3LΔ7C). As disclosed in the above-captioned patent application, the mutations encompassed by the present invention are those which decrease, but do not abolish, binding of the mutant E3L gene product to dsRNA relative to the native E3L gene product.

3. I am a co-author of Brandt et al, 2001, *J. Virol.* 75(2):850-85 (hereinafter "Brandt") and thoroughly familiar with the disclosures therein. Brandt discloses a recombinant vaccinia virus lacking E3L (hereinafter "VVΔE3L") and a recombinant vaccinia virus comprising a truncated E3L gene that lacks nucleic acids encoding the carboxy terminal 26 amino acids (hereinafter "VVE3LΔ26C"). Brandt does not disclose whether the E3LΔ26C gene product binds dsRNA.

4. In accordance with the invention, in order to determine whether the dsRNA binding affinities of the E3LΔ7C gene product, the E3LΔ26C gene product, and wild type E3L gene product differ, respective preparations of total E3L protein were prepared by *in vitro* transcription and translation (paragraph 5 below), exposed to dsRNA (paragraph 6 below), and formation of nucleoprotein complexes was detected (paragraph 7 below).

5. Five micrograms of pBluescript or pGEM-5T plasmids containing wild type or mutant E3L DNA were linearized with HindIII, and T7 RNA polymerase (Promega) was used according to the manufacturer's specifications to transcribe RNA. After completion of the transcription reaction, samples were treated with DNase and extracted with phenol chloroform and chloroform. RNA was then precipitated from the samples with ethanol and resuspended in 20 μl of diethylpyrocarbonate-treated water. Aliquots of RNA (10 μl) were used in *in vitro* translation reactions (Promega). The translation reaction mixture contained 35 μl of nuclease-

treated rabbit reticulocyte lysate, 20 units of RNasin, 20 μM amino acid mixture minus methionine, and 50 μCi of [^{35}S]methionine (1186 Ci/mmol; 1 Ci = 37 GBq). The reactions were carried out at 30° for 1.5 hr. After completion of the reaction, 15 μl of the mixture was bound to poly(rl) poly(rC) - agarose.

6. Poly(rl) poly(rC)-agarose or reovirus dsRNA-agarose (Langland *et al.*, 1992) was washed three times in buffer A (150 mM KCl, 20mM HEPES, pH 7.5, 10% glycerol, 5mM MgOAc, 1mMDTT, 1mM benzamidine). [^{35}S]methionine-labeled *in vitro* translation mixtures were added to the washed poly(rl) poly(rC)-agarose and incubated at 4° with occasional mixing for 1 hr. The agarose was then washed three times in buffer A. dsRNA-binding proteins were eluted from the agarose by adding an equal volume of 2X SDS/PAGE sample buffer and boiling for 3min. Proteins were separated by SDS/PAGE and visualized by autoradiography.

7. Results for this study are depicted in Figure 1 below.

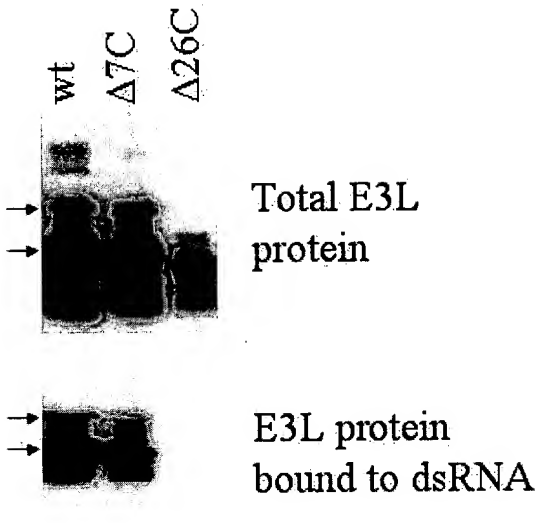


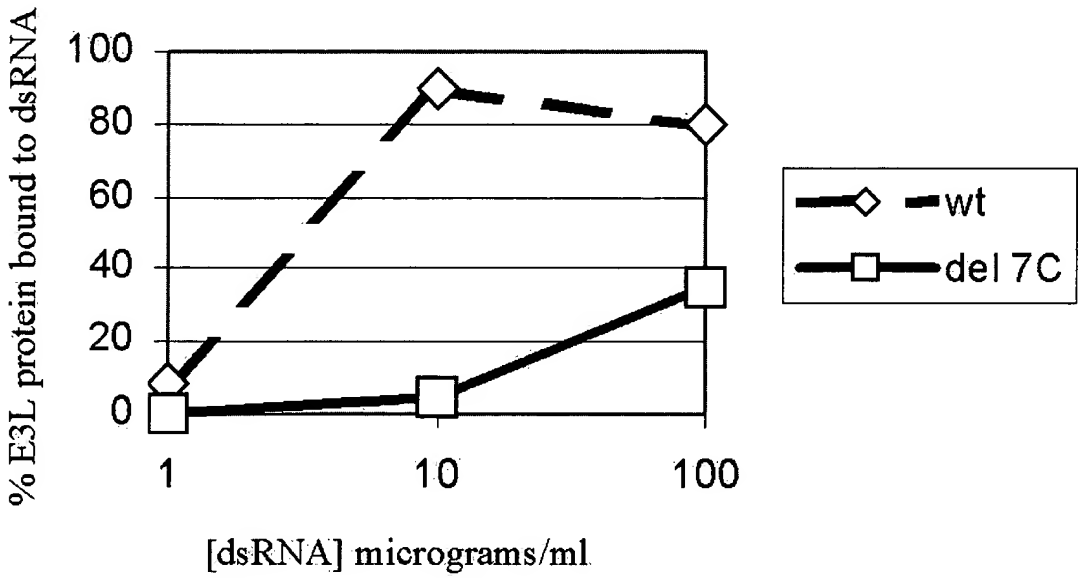
Figure 1. Vaccinia Virus E3L proteins bound to dsRNA

8. The upper panel of Figure 1 shows the total E3L protein produced by the respective E3L genes (arrows). The lower panel confirms prior observations that wild-type E3L protein binds dsRNA (left lane). Similarly, E3LΔ7C protein also binds dsRNA, albeit with a slightly reduced affinity (center lane). By contrast, E3LΔ26C protein did not form detectable nucleoprotein complexes when exposed to dsRNA (right lane).

9. The relative dsRNA-binding affinity of wild type E3L, the E3LΔ26C of Brandt, and the E3LΔ7C of the invention were tested. Increasing concentrations of soluble poly(rI) poly(rC) (0 to 100 μg/ml) were incubated for 1 hr on ice with [^{35}S]methionine-labeled in vitro translation mixtures. Buffer A washed poly(rI) poly(rC)-agarose was then added to the mixture and incubated on ice for 1 hr. with occasional mixing. The agarose was then washed

three times in buffer A. Proteins were eluted from the agarose by adding an equal volume of 2X SDS/PAGE sample buffer and boiling for 3 min. Proteins were separated by SDS/PAGE and visualized by autoradiography. The relative amount of E3L proteins bound to poly(rl) poly(rC)-agarose were determined by scanning densitometry of the autoradiogram. The maximal binding of E3L proteins to poly(rl) and poly(rC)-agarose was in the presence of no soluble poly(rl) poly(rC) and was given the arbitrary value of 100%. The relative amount of E3L proteins bound to soluble (poly(rl) poly(rC) was obtained by subtracting the value of E3L proteins bound to poly(rl) poly(rC)-agarose from 100%.

10. Results for this study are depicted in Figure 2 below.



11. Figure 2 shows the percentage of E3L protein bound to dsRNA as a function of the concentration of the dsRNA-binding solution. No data points for E3LΔ26C were plotted since no dsRNA binding could be detected at any concentration tested (i.e. 0-100 μg/ml).

However, as demonstrated, the E3LΔ7C protein binds to dsRNA although with a lower affinity than wild type protein.

12. In conclusion, under the assay conditions used herein, there is a qualitative difference in the dsRNA binding properties of wild-type E3L protein and E3LΔ7C protein compared with E3LΔ26C protein. Specifically, both wild-type E3L protein and E3LΔ7C protein bind dsRNA while E3LΔ26C protein does not.

13. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of any patent issuing from the above-captioned patent application.

10/6/04
Date

BERTRAM JACOBS

Enclosure

CURRICULUM VITAE

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EDUCATION

Ph. D., Biochemistry, June, 1981
University of California, Berkeley
Dissertation advisor: E.E. Penhoet
Dissertation title: "The mechanism of assembly of vesicular stomatitis virus."

B.S., Biology, June, 1974
Rutgers University

Johns Hopkins University, 1968-1971

PROFESSIONAL EXPERIENCE

7/00-6/01	Distinguished Visiting Professor Centro Nacional Biotechnologia, Madrid, Spain Aventis, Pasteur, Toronto, Canada Department of Microbiology and Molecular Genetics University of Florida
8/96-7/00	Director Graduate Degree Program in Molecular and Cellular Biology Arizona State University
8/96-present	Professor Department of Microbiology Arizona State University
8/90-8/96	Associate Professor Department of Microbiology Arizona State University

6/91-1/92	Visiting Associate Research Professor Molecular Mechanisms of Carcinogenesis Laboratory Human Retrovirus Section National Cancer Institute Frederick Cancer Research Facility
8/85-3/90	Assistant Professor Department of Microbiology Arizona State University
6/84-8/85	Post-Doctoral Research Associate Department of Biological Sciences Section of Biochemistry and Molecular Biology University of California, Santa Barbara
1/81-6/84	Lecturer (50% time)/Post-Doctoral Research Associate (50% time) Department of Biological Sciences Section of Biochemistry and Molecular Biology University of California, Santa Barbara
9/75-1/81	NIH Pre-Doctoral Fellow Department of Biochemistry University of California, Berkeley
8/74-8/75	Research Assistant Department of Pathology Cornell University Medical College

PUBLICATIONS

1. Lasky SR, Jacobs BL, Samuel CE. Mechanism of interferon action. Characterization of sites of phosphorylation in the interferon-induced phosphoprotein P1 from mouse fibroblasts: evidence for two forms of P1. *J Biol Chem.* 1982;257:11087-93.
2. Jacobs BL, Penhoet EE. Assembly of vesicular stomatitis virus: distribution of the glycoprotein on the surface of infected cells. *J Virol.* 1982;44:1047-55.
3. Miyamoto NG, Jacobs BL, Samuel CE. Mechanism of interferon action. Effect of double-stranded RNA and the 5'-O-monophosphate form of 2',5'-oligoadenylate on the inhibition of reovirus mRNA translation in vitro. *J Biol Chem.* 1983;258:15232-7.
4. Jacobs BL, Samuel CE. Biosynthesis of reovirus-specified polypeptides: the reovirus s1 mRNA encodes two primary translation products. *Virology.* 1985;143:63-74.
5. Jacobs BL, Atwater JA, Munemitsu SM, Samuel CE. Biosynthesis of reovirus-specified polypeptides. The s1 mRNA synthesized in vivo is structurally and functionally indistinguishable from in vitro-synthesized s1 mRNA and encodes two polypeptides, sigma 1a and sigma 1bNS. *Virology.* 1985;147:9-18.

6. Jacobs BL, Miyamoto NG, Samuel CE. Mechanism of interferon action: studies on the activation of protein phosphorylation and the inhibition of translation in cell-free systems. *J Interferon Res.* 1988;8:617-31.
7. Imani F, Jacobs BL. Inhibitory activity for the interferon-induced protein kinase is associated with the reovirus serotype 1 sigma 3 protein. *Proc Natl Acad Sci U S A.* 1988;85:7887-91.
8. Jacobs BL, Imani F. Histone proteins inhibit activation of the interferon-induced protein kinase by binding to double-stranded RNA. *J Interferon Res.* 1988;8:821-30.
9. Jacobs BL, Ferguson RE. The Lang strain of reovirus serotype 1 and the Dearing strain of reovirus serotype 3 differ in their sensitivities to beta interferon. *J Virol.* 1991;65:5102-4.
10. Watson JC, Chang HW, Jacobs BL. Characterization of a vaccinia virus-encoded double-stranded RNA- binding protein that may be involved in inhibition of the double- stranded RNA-dependent protein kinase. *Virology.* 1991;185:206-16.
11. Langland JO, Jacobs BL. Cytosolic double-stranded RNA-dependent protein kinase is likely a dimer of partially phosphorylated Mr = 66,000 subunits. *J Biol Chem.* 1992;267:10729-36.
12. Chang HW, Watson JC, Jacobs BL. The E3L gene of vaccinia virus encodes an inhibitor of the interferon- induced, double-stranded RNA-dependent protein kinase. *Proc Natl Acad Sci U S A.* 1992;89:4825-9.
13. Lyubchenko YL, Jacobs BL, Lindsay SM. Atomic force microscopy of reovirus dsRNA: a routine technique for length measurements. *Nucleic Acids Res.* 1992;20:3983-6.
14. Lyubchenko YL, Gall AA, Shlyakhtenko LS, et al. Atomic force microscopy imaging of double stranded DNA and RNA. *J Biomol Struct Dyn.* 1992;10:589-606.
15. Clemens, M., Hershey, J.W.B., Hovanessian, A.G., Jacobs, B.L., Katze, M.G., Kaufman, R., Lengyel, P., Samuel, C.E., Sen, G, and Williams, B.R.G. (1993). PKR: proposed nomenclature for RNA-dependent protein kinase induced by interferon. *J. Interferon Res* **13**:241.
16. Davies MV, Chang HW, Jacobs BL, Kaufman RJ. The E3L and K3L vaccinia virus gene products stimulate translation through inhibition of the double-stranded RNA-dependent protein kinase by different mechanisms. *J Virol.* 1993;67:1688-92.
17. Chang HW, Jacobs BL. Identification of a conserved motif that is necessary for binding of the vaccinia virus E3L gene products to double-stranded RNA. *Virology.* 1993;194:537-47.
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20. Langland JO, Pettiford S, Jiang B, Jacobs BL. Products of the porcine group C rotavirus NSP3 gene bind specifically to double-stranded RNA and inhibit activation of the interferon-induced protein kinase PKR. *J Virol.* 1994;68:3821-9.
21. Denzler KL, Jacobs BL. Site-directed mutagenic analysis of reovirus sigma 3 protein binding to dsRNA. *Virology.* 1994;204:190-9.

22. Trelease RN, Choe SM, Jacobs BL. Conservative amino acid substitutions of the C-terminal tripeptide (Ala- Arg-Met) on cottonseed isocitrate lyase preserve import in vivo into mammalian cell peroxisomes. *Eur J Cell Biol.* 1994;65:269-79.
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35. Jacobs BL, Langland JO. Reovirus sigma 3 protein: dsRNA binding and inhibition of RNA-activated protein kinase [In Process Citation]. *Curr Top Microbiol Immunol.* 1998;233 REOVIR.I:185-96.
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42. Jacobs, B.L. (2000). Translational Control in Poxvirus-infected Cells. In, *Translational Control of Gene Expression*, CSHL Press,2000.
43. Brandt, T. A. and B. L. Jacobs (2001). "Both carboxy- and amino-terminal domains of the vaccinia virus interferon resistance gene, E3L, are required for pathogenesis in a mouse model." J Virol **75**(2): 850-6.
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46. Jancovich, J. K., E. W. Davids, A. Seiler, B. L. Jacobs and J. P. Collins (2001). "Transmission of the Ambystoma tigrinum virus to alternative hosts." Dis Aquat Organ **46**(3): 159-63.
47. He, Y., H. Nakao, S. L. Tan, S. J. Polyak, P. Neddermann, S. Vijaysri, B. L. Jacobs and M. G. Katze (2002). "Subversion of cell signaling pathways by hepatitis C virus nonstructural 5A protein via interaction with Grb2 and P85 phosphatidylinositol 3-kinase." J Virol **76**(18): 9207-17.
48. Langland, J. and B. Jacobs (2002). "The Role of the PKR-Inhibitory Genes, E3L and K3L, in Determining Vaccinia Virus Host Range." Virology **299**(1): 133.
49. Xiang, Y., R. C. Condit, S. Vijaysri, B. Jacobs, B. R. Williams and R. H. Silverman (2002). "Blockade of interferon induction and action by the E3L double-stranded RNA binding proteins of vaccinia virus." J Virol **76**(10): 5251-9.
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51. Vijaysri, S., Talasela, L., Mercer, A.A., Mcinnes, C.J., Jacobs, B.L., Langland, J.O. (2003). The ORF virus E3L homologue is able to complement deletion of the vaccinia virus E3L gene *in vitro* but not *in vivo*. *Virology*, in press.

EXTRAMURAL RESEARCH SUPPORT

NIH/NIAID 1 U01 AI057303-01, July, 2003-2008, $1,250,000/year entire project.
"Development of a Safer Smallpox Vaccine" Pending

NIH/NIAID R01 AI052347-01, July, 20023-2008, $275,000 direct costs/year. "Role of the E3L and K3L genes in poxvirus pathogenesis" Pending.

NIH/NIAID R21 AI052347-01, July, 2002-2003, $200,000 direct costs/year. "Role of the E3L gene in poxvirus pathogenesis"

NIH/NIAID R21 AI053457-01, July, 2002-2004, $150,000/year, direct costs. "An Improved Vaccine for Protection Against Smallpox"

NIH/NIAID R21 AI52787-01, July, 2002-2004, $150,000/year, direct costs. "Mucosal Vaccination With A Vaccinia Virus-Based Vector"

Dana Foundation, October 2001-2006, $50,000/year total costs. "Pathogenic Mechanisms in Poxviruses"

Wallace Foundation, December 2000-2003. $100,000/year total costs. "Development of Viruses to Treat Cancer."

Arizona Disease Control Research Commission, July,1, 1999-June 30, 2002. $450,000. " Anti-sense mediated induction of apoptosis in cancer cells," PI.

Arizona Disease Control Research Commission, July,1, 1999-June 30, 2001. $500,000. "Viral vectors for treatment of brain cancer," Co-PI.

Arizona Disease Control Research Commission, July 1, 1995-June 30, 1998. $90,000. "Regulation of Programmed Cell Death in human cancer cells." #6-027. Principal investigator.

American Cancer Society; 7/1/95-6/30/97. $180,000. "Mechanism of transformation by inhibitors of the interferon system." VM-151. Principal investigator.

National Institutes of Health; 4/1/95-3/31/99; $950,000. "Role of E3L in poxvirus replication/IFN-resistance." R01 CA48654. Principal investigator. No-cost renewal 3/31/91-3/1/00.

National Institutes of Health; 10/91-10/94; $555,296. "Interaction of anti-HIV drugs with placental models." R01 AI32316. Co-principal Investigator.

National Institutes of Health; 4/91-4/94; $411,971. "Mechanism of action of interferon against HIV." R01 AI30349. Principal Investigator.

National Institutes of Health; 5/88-5/93; $493,369. "Control of the interferon-induced protein kinase." R29 CA48654. Principal Investigator.

Arizona Disease Control Research Commission; 7/87-7/90; $74,635. "Mechanism of induction of virus associated diabetes mellitus." Principal Investigator.

American Diabetes Association; 7/87-7/90; $69,710. "Mechanism of induction of virus associated diabetes mellitus." Principal Investigator.

National Institutes of Health; 9/89-9/90; $22,033. "Small Instrumentation Grant Program." Co-principal Investigator.

INTRAMURAL RESEARCH SUPPORT

Arizona State University Faculty Grant-in-aid; 1/95-12/95; $6,000. "Regulation of programmed cell death in human cancer cells."

Arizona State University Biomedical Research Fund; 1/89-1/90; $8,413. "Characterization of a putative vaccinia virus inhibitor of interferon action."

Arizona State University Biomedical Research Fund; 1/88-1/89; $7,364. "Characterization of interferon-resistant viruses."

Arizona State University Biomedical Research Fund; 1/87-1/88; $8,500. "The role of interferon in the etiology of insulin dependent diabetes mellitus."
Arizona State University Faculty Grant-in-aid; 1/87-1/88; $3,000. "Molecular cloning of cDNA for an interferon-induced mRNA."
Arizona State University Research Fund; 1/86-1/87; $8,740. "Purification of enzymes involved in the antitumor and antiviral effects of interferon."
Arizona State University Faculty Grant-in-aid; 1/86-1/87; $3,000. "Molecular cloning of cDNA for an interferon-induced mRNA."

ASSOCIATIONS

American Society for Virology
American Society for Microbiology
American Association for the Advancement of Science

HONORS, EDITORIAL BOARDS, PROPOSAL REVIEW, etc.

Ad-hoc member, Virology Study Section, NIH, June, 2003
Invited Speaker, NIH Poxvirus Bioterrorism Research Meeting, Bethesda, MD. April, 2003. "Replication competent, attenuated strains of vaccinia virus as improved vaccines." and "Role of a Z-DNA binding protein in poxvirus pathogenesis."
Arizona/Nevada Branch ASM, Keynote Speaker, February, 2003
University of Illinois 6th Annual Conference on New and Re-emerging Infectious Diseases, Keynote Speaker, April ,2003.
Invited Speaker, NIH Poxvirus Bioterrorism Research Meeting, Bethesda, MD. May 15-17, 2002. "Replication competent, attenuated strains of vaccinia virus as improved vaccines."
Member, US Inter-agency Smallpox Working Group, 2002
Member, Poxvirus Proteomics Review Panel, NIH, Dec. 2002
Ad-hoc member, Experimental Virology Study Section, NIH, Oct., 2002
Poxvirus expert, Biotechnology Enhancement Program Site Visit Team, VECTOR, Novosibirsk, Russia. July 20-25, 2002. Site visit is to audit a joint CDC/VECTOR grant, "Variola (smallpox) Genome Project."
Consultant, ISTC Project 1987, VECTOR, January 2003-
Member, Rapid Response to Bioterrorism Study Section, NIH, June, 2002
Chair, Poxvirus Pathogenesis Session of the International Poxvirus Symposium, Lake Placid, NY September 19-25, 2002.
Associate Editor, *Virology*, 1993-2002
Ad hoc reviewer for NSF, USDA, Canadian MRC, Journal of Biological Chemistry, Molecular and Cellular Biology, Virus Research, the Wellcome Trust.

RECENT INVITED LECTURES/SEMINARS

2003
ASU Sigma Xi Banquet, Keynote Speaker
ASU President's Community Enrichment Program, Journeys of the Mind
Department of Microbiology, St. Louis University

Arizona/Nevada Branch ASM, Keynote Speaker
University of Illinois 6th Annual Conference on New and Re-emerging Infectious Diseases, Keynote Speaker
2002
USAMRIID
Chair, Poxvirus Pathogenesis Workshop, International Poxvirus Symposium
NIH Poxvirus Research Meeting
Department of Chemistry, NAU
Department of Microbiology, University of Washington
Institute for Molecular Virology, Munich, Germany
Department of Microbiology and Immunology, University of Arizona School of Medicine
2001
Department of Microbiology, SUNY, Buffalo
Department of Cancer Biology, Cleveland Clinic
Department of Biology, MIT
Department of Microbiology, University of Western Ontario
Department of Microbiology and Molecular Genetics, University of Florida
2000
Centro Nacional Biotechnologia

TEACHING

Arizona State University, 1985-present

Honors: Nominated for College of Liberal Arts and Sciences Distinguished Teaching Award (1985/86, 1986/87, 1988/899, 1990/91). Selected for College of Liberal Arts and Sciences Distinguished Teaching Award, 1990/91. Graduate Student Council Outstanding Faculty Mentor, 1996.

MIC/PGS/JUS 394	HIV Disease in America. 3 Credits. Fall and Spring (1997-present). Lecturer and Faculty-in-charge. Class size, 70-80 students.
MIC 498	Molecular Techniques. 3 credits, Spring Semester (1991-2002). Team taught. Lecturer and Faculty-in-charge. Class size 6-12 students.

MIC 485	General Virology. 3 credits, Fall Semester (1985-2002). Sole lecturer and Faculty-in-charge. Class size 30-40.
MIC 486	Virology Laboratory. 2 sections, 2 credits each, Fall Semester (1985-1989). Faculty-in-charge. Class size, appr. 10 students per section.
MIC 494/598	ST: Topics in Molecular Virology. 3 credits, Spring Semester (1986-1991). Faculty-in-charge. Class size 10-15.
MIC 591	S: Virology. 1 credit, Fall and Spring Semesters (1989-present). Faculty-in-charge. Class size 5-10.
MIC 591	S: Molecular and Cellular Biology. 1 credit, Fall and Spring Semesters (1986-present). Team taught. Class size 5-10.
MIC 591	S: Genetics. 1 credit, Fall and Spring Semesters (1985-2000). Team taught (with Zoology faculty). Class size 10-15.
MIC 591	S: Enzymology. 1 credit, Fall and Spring Semesters (1986-1989). Team taught. Class size 5-10.
MCB 556	Advanced Molecular and Cellular Biology. 3 credits, Spring Semester (1993, 1995). Team Taught. Class size 15-20.

University of California, Santa Barbara, 1981-1984

BIO 11A	Introductory Biology. 4 credits, Fall Semester (1982-1984). Team taught. Class size 300-400.
BIO 122	Virology. 3 credits, Spring Semester (1981-1984). Faculty-in-charge and sole lecturer. Class size 70-75.

University of California, Berkeley, 1980

BCM 102	General Biochemistry. 4 credits, Summer (1980). Faculty-in-charge and sole lecturer. Class size 50.
	Organic Chemistry review for students taking MCAT and GRE exams. Faculty-in-charge and sole lecturer. Class size 20.

EXHIBIT A

GRADUATE COMMITTEES CHAIRED

NAME	DEGREE	CURRENT POSITION
Imani, Farhad	Ph.D., 8/89	Research Associate, The Johns Hopkins University School of Medicine, Baltimore, MD
Shearer, Michael	M.N.S., 6/87	Research Assistant, Southwest Biomedical Research Foundation, San Antonio, Texas
Watson, Julia	Ph.D., 12/90	Research Associate, Ribogene, Inc, Hayward, CA
Langland, Jeffrey	Ph.D., 12/90	Research Assistant Professor, Arizona State University
Chang, Cathy	Ph.D., 6/93	Research Scientist, Stratagene
Denzler, Karen	Ph.D., 6/94	Postdoctoral Research Associate, Mayo Clinic, Scottsdale
Shors, Scott	Ph.D., 6/94	Postdoctoral Research Associate, NIH, Bethesda, MD
Shors, Terri	Ph.D., 6/95	Assistant Professor, Department of Biology and Microbiology, University of Wisconsin, Oshkosh
Kibler, Karen	Ph.D., 12/97	Post-doctoral research associate, Mayo Clinic, Scottsdale
Zeman, Cameron	M.S., 12/97	Medical School, University of Iowa
Matt Banaszak	M.S., 12/98	Technician, VA Medical Center, Phoenix
Jennifer Biesterfeldt	M.S., 12/98	Technician, Mayo Clinic, Scottsdale
Kim Perkins	Ph.D., 12/99	Post-doctoral research associate, Mayo Clinic, Scottsdale
Sangeetha Vijaysri	Ph.D., 8/01	Industry
Teresa Brandt	Ph.D., 2001	Industry
Maneesha Muralinath	Ph.D., 2003	
Stacy Frederick	Ph.D. candidate	
Kelly Trainor	Ph.D. candidate	
Chandra Mitnik	Ph.D. candidate	

Kevin Hauns	Ph.D. candidate
Mat Pearcy	Ph.D. candidate
Garalyn Gentarra	Ph.D. candidate